

**The Geometry of Trauma:
Identifiability and Reachability in Models of Psychological
Recovery**

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Abstract

Recovery after trauma is increasingly modelled as movement through a state space with stable symptom states, thresholds and interventions that push a person between them. We ask which conclusions such models support independently of arbitrary modelling choices, and which can be identified from clinical data. In the canonical bistable model, support delivered at a bounded rate U moves a person from the low state to the recovered state exactly when U exceeds the largest opposing drift, $U_c = 0.3849$. Near that threshold the passage time diverges as $(U - U_c)^{-1/2}$: support 1% above threshold needs a dose of 14.1, against 1.55 at twice the threshold. Under quadratic cost the best adaptive schedule spends 4 times the potential barrier, and the best constant schedule spends 1.142 times that. With noise the threshold becomes a probability: support at half the threshold raises recovery by a fixed horizon about a hundredfold, as the Kramers rate predicts. When support varies across people, initial severity predicts recovery with an area under the curve of 0.60, since a mild case below the accessible boundary cannot recover and a severe case above threshold can. Untreated data leave the effect of support unidentified. Under confounding by indication the estimated effect can change sign, a true gain of 0.3 appearing as -0.50 , and a conclusion that support suffices tolerates confounding of at most 0.230 in the gain. A randomized trial of 593 participants reaches that conclusion with probability 0.80. Euclidean distance to recovery depends on the units of the state: its area under the curve ranges from 0.63 to 0.96 across unit choices, while reachability and hitting times do not change.

Introduction

Most people exposed to a potentially traumatic event do not develop lasting disorder. Across 54 studies of trajectories after such events, resilience was the modal course, averaging 65.7% of samples, followed by recovery at 20.8%, chronic symptoms at 10.6% and delayed onset at 8.9% (Galatzer-Levy et al. 2018; see also Bonanno 2004; Bonanno et al. 2023). The same exposure can therefore lead to very different courses, and severity at presentation predicts course imperfectly (Bryant 2019). Dynamical accounts explain this heterogeneity by treating symptoms as a system with more than one stable state. Psychopathology has been described as a network of mutually reinforcing symptoms (Borsboom 2017; Cramer et al. 2016), resilience as the dampening of such networks by protective factors (Kalisch et al. 2019), and transitions into and out of disorder as tipping points preceded by critical slowing down (Scheffer et al. 2009; van de Leemput et al. 2014). Change in psychotherapy is often discontinuous (Hayes et al. 2007). For post-traumatic stress disorder specifically, a minimal model with an autocatalytic symptom driver is bistable, and a single resilience parameter below a threshold produces a persistent switch to a high-symptom state, with widely varying recovery times near the threshold explained by a dynamical ghost (Grosskopf et al. 2026).

A second line of work treats treatment as control. Control-theoretic statistics have been proposed for selecting intervention targets in symptom networks (Henry et al. 2022), network control theory has been used to predict the effect of a psychological intervention (Stocker et al. 2023), and the same framework has been proposed for personalizing therapy (Hofmann 2025). Person-specific

network models of post-traumatic stress have been estimated from intensive longitudinal data (Hertz-Palmor et al. 2025).

These models invite a geometric description of recovery, with distances to recovery, barriers, paths and landscapes. Three questions determine what that description is worth. Which quantities follow from the dynamics and which depend on how the state is coordinatised? What does a bounded capacity to deliver support imply for whether and how quickly a person can reach recovery? And which of these quantities can be identified from the data that clinical services generate, in which support is given to those who appear to need it? We answer all three in the simplest model that has the relevant structure. Every number below is a property of a stated model and none is an estimate about patients.

Post-traumatic growth is outside the scope of the claims. Growth after adversity is a distinct and multidimensional outcome (Tedeschi and Calhoun 2004), retrospective self-reports of growth are largely unrelated to measured change from before to after trauma (Frazier et al. 2009), and the evidence that moderate adversity is followed by better outcomes comes from associations in longitudinal samples (Seery et al. 2010), which do not establish that adversity caused the difference.

Model

A scalar coordinate x summarises recovery, with larger values better. It evolves as

$$dx = (f(x) + bu + e) dt + \sigma dW, \quad f(x) = x - x^3,$$

where $u(t) \in [0, U]$ is support delivered at a rate bounded by U , b is its causal gain, e is a sustained environmental input and W is a Wiener process. With $u = e = \sigma = 0$ the model has stable equilibria at $x = -1$, the persistent symptomatic state, and $x = +1$, the recovered state, separated by an unstable equilibrium at $x = 0$. The drift is the negative gradient of the potential $V(x) = x^4/4 - x^2/2$, with a barrier $\Delta V = V(0) - V(-1) = 0.25$. Unless stated otherwise $b = 1$ and $e = 0$. The one-dimensional reduction is deliberate: every result below concerns a property that a richer model inherits along the direction in which it escapes from the symptomatic state.

A threshold for bounded support

On the way from -1 to 0 the dynamics oppose recovery with drift $-f(x)$, which is largest at the bottleneck $x_c = -1/\sqrt{3}$, where it equals

$$U_c = \frac{2}{3\sqrt{3}}.$$

Constant support $U > U_c$ carries a person from $x = -1$ past $x = 0$ in finite time, after which the unforced dynamics complete the recovery, so support can be withdrawn once the state has crossed

the separatrix. Support $U < U_c$ cannot, however long it is given. The threshold is $U_c = 0.3849$, which a direct maximisation of $-f$ over a grid of 200,001 points reproduces to 1.3e-13 (Figure 1a).

Just above threshold the dynamics pass slowly through the region where the opposing drift nearly cancels the support. The passage time from -1 to 0 is

$$\tau(U) = \int_{-1}^0 \frac{dx}{f(x) + U},$$

and near the threshold the integral is dominated by the bottleneck, where $f(x) + U \approx (U - U_c) - \sqrt{3}(x - x_c)^2$, giving $\tau(U) \approx \pi / \sqrt{\sqrt{3}(U - U_c)}$ (Strogatz 2018). At $U - U_c = 10^{-4}$ the exact passage time is 0.991 of this asymptote, and a fit over the three points closest to threshold gives an exponent of -0.518 against the asymptotic -0.5 (Figure 1b). Support 1% above threshold needs a passage time of 36.4, 10% above needs 10.0, and twice the threshold needs 2.02. The quadrature agrees with a fourth-order Runge–Kutta integration to a relative error of 2.9e-10. This is the ghost that produces broad recovery timescales in the PTSD model of Grosskopf et al. (2026), seen from the side of the intervention.

The dose of support, $\int_0^\tau u dt$, can be written as $\int u/(f(x) + u) dx$ along the path. On $(-1, 0)$, where $f < 0$, the integrand decreases in u , so for a given bound U the smallest dose is obtained by delivering the bound throughout, and that dose is $U\tau(U)$. It decreases with intensity: 14.1 at 1% above threshold, 4.25 at 10% above and 1.55 at twice the threshold, approaching 1 only as intensity grows without bound (Figure 1c). Support just above threshold is therefore both slow and wasteful in this model, because most of it is spent holding the state near the bottleneck.

If the cost of support grows with the square of its intensity, the effort $\int u^2 dt = \int u^2/(f + u) dx$ is minimised pointwise at $u^*(x) = -2f(x)$, twice the opposing drift at each point, which peaks at $2U_c = 0.770$ at the bottleneck. The minimal effort is $\int_{-1}^0 4|f(x)| dx = 4\Delta V = 1$, the relation between control effort and potential barriers familiar from the theory of large deviations (Freidlin and Wentzell 2012). The best constant schedule, at $U = 0.620$ or 1.61 times the threshold, costs 1.142 times the adaptive minimum.

A sustained environmental input e plays a different role from a transient push. An input below threshold, such as $e = 0.3$, moves the symptomatic equilibrium from -1 to -0.786 without removing it, and the state returns to -1 when the input ends. An input or support above threshold removes the equilibrium, and a state that has crossed $x = 0$ stays recovered when it ends. Changing the landscape and moving the state through it are distinct interventions with distinct persistence.

Noise and the probability of recovery

With noise the threshold becomes a probability. Simulations of 20,000 paths over a horizon of 50 time units, counting recovery as reaching $x = 0.5$, show how support below threshold changes it (Figure 2b). With noise amplitude $\sigma = 0.25$, the probability of recovery by the horizon is 0.0039

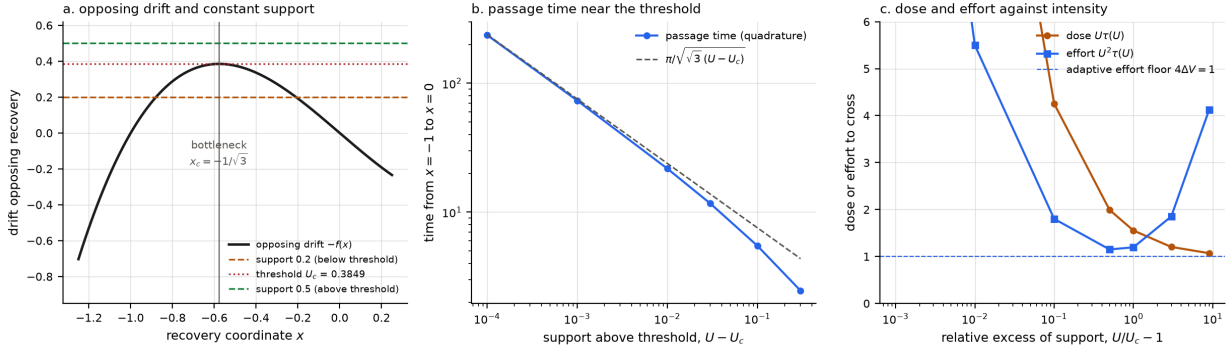


Figure 1: The threshold for bounded support. (a) The drift opposing recovery on the way from the symptomatic to the recovered state, with support below, at and above the threshold U_c . (b) Passage time from $x = -1$ to $x = 0$ against the excess of support over threshold, with the saddle-node asymptote. (c) Dose and quadratic effort needed to cross against the relative excess of support; the dashed line is the effort of the best adaptive schedule, $4\Delta V = 1$.

without support, 0.40 with support 0.2, about half the threshold, and 0.93 with support 0.3. Support at half the threshold therefore raises the probability of recovery about 105-fold. The reason is that support tilts the potential and lowers the barrier, from 0.25 without support to 0.025 at support 0.3, and escape rates depend exponentially on the barrier divided by the noise intensity (Kramers 1940). Where recovery within the horizon is rare, below probability 0.3, the Kramers approximation agrees with the simulations to within 14%. With $\sigma = 0.35$ the probability without support is already 0.15. Whether a given support level is “enough” is therefore a statement about a probability at a horizon, which depends on the noise and cannot be read from the deterministic threshold alone. The appropriate formal object is probabilistic reachability, the probability of reaching a target set while remaining in a safe set under an admissible policy (Abate et al. 2008).

Accessible states and the information in severity

Below threshold, constant support U reaches recovery only from states above the middle root $s(U)$ of $x - x^3 + U = 0$; states below it settle at the lower root. The boundary moves from $s = 0$ without support to $s = -0.209$ at support 0.2, and every state becomes accessible at U_c (Figure 2a). Two illustrative cases show why severity at presentation is a poor guide. Case A starts deep in the symptomatic region at $x_0 = -1.4$ with support 0.45, above threshold, and reaches $x = 0.5$ after 8.5 time units. Case B starts at $x_0 = -0.3$, much milder, with support 0.2, below threshold; since $-0.3 < s(0.2) = -0.209$, it never recovers. Above threshold, the starting point matters little because the bottleneck dominates the passage time: with support 0.40, starting from -1.5 takes 17.8 time units and starting from -0.8 takes 16.3, a ratio of 1.09.

In a synthetic population of 4,000 in which initial state and support are drawn independently, with 0.567 recovering by a horizon of 20, initial severity alone discriminates those who recover from those who do not with an area under the receiver operating characteristic curve (AUROC) of 0.60. The support bound alone reaches 0.97. The model’s own margin, support minus the largest

opposing drift on the remaining path, reaches 0.9999, which it does by construction. The comparison does not show that severity is uninformative in any clinical population. It shows that when the capacity to receive effective support varies across the threshold, severity at presentation carries little of the information that determines recovery, and that the incremental value of accessibility over severity is the quantity a longitudinal study would need to estimate.

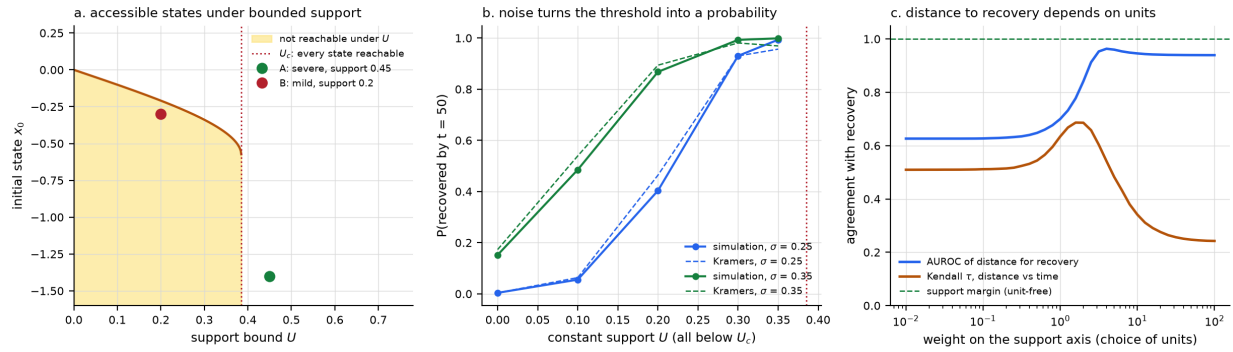


Figure 2: Accessibility, noise and coordinates. (a) States from which constant support U reaches recovery; the boundary is the middle root $s(U)$, and above U_c every state is reachable. Case A is severe with support above threshold; case B is mild with support below it. (b) Probability of recovery by $t = 50$ against support below threshold, from simulation and from the Kramers rate, for two noise levels. (c) Agreement of Euclidean distance to recovery with recovery and with hitting time, against the weight placed on the support axis, compared with the unit-free support margin.

What clinical data can identify

The support gain b has so far been assumed known. In practice it must be estimated, and three results bound what can be learned.

Untreated data do not identify it at all. In the discrete-time model $x_{t+1} = ax_t + bu_t + \varepsilon_t$ with $u_t = 0$ throughout, the likelihood does not involve b : models with $b = +0.5$ and $b = -0.5$ driven by the same noise produce identical trajectories, with a largest absolute difference of 0.0 over 10,000 steps. No amount of untreated observation distinguishes a helpful intervention from a harmful one.

Observational data in which support is given to those who appear to need it are confounded by indication (Salas et al. 1999). Let an unobserved hazard h , such as ongoing threat, lower recovery with coefficient $c < 0$ and raise the support delivered, $u = \gamma h + \nu$, with h and ν of standard deviations σ_h and σ_ν . Regressing x_{t+1} on x_t and u_t then estimates

$$\hat{b} \rightarrow b + \frac{c \gamma \sigma_h^2}{\gamma^2 \sigma_h^2 + \sigma_\nu^2},$$

whose bias is largest in magnitude, $|c| \sigma_h / (2\sigma_\nu)$, when $\gamma = \sigma_\nu / \sigma_h$. The estimated sign is reversed whenever the bias exceeds the true gain, which for $\sigma_h = 1$ happens on the interval of γ where $b\gamma^2 - |c|\gamma + b\sigma_\nu^2 < 0$. With $b = 0.3$, $c = -0.8$ and $\sigma_\nu = 0.5$ the largest bias is 0.8, the sign is reversed for γ between 0.097 and 2.57, and at $\gamma = 0.5$ a helpful intervention with gain 0.3 is estimated at -0.50 . Simulations with 200,000 observations agree with the closed form to within 0.0028 (Figure 3a). Adjusting for the observed state does not remove this bias, because the hazard is unobserved; methods that address time-varying confounding require the confounders to be measured (Robins et al. 2000).

A conclusion robust to this uncertainty must hold over every gain in the identified set (Manski 2003). In the bistable model, support of bound $U = 0.5$ reaches recovery from the symptomatic state if and only if $bU > U_c$, that is, if the gain exceeds 0.770. If the true gain is 1 and observational data identify it only up to a confounding bias of magnitude G , the conclusion that support of this intensity suffices holds for every gain in the identified set only if G is below 0.230. The largest bias in the confounding example is 3.5 times that margin. A randomized trial removes the confounding: with two equal arms receiving $u = 0$ or $u = U$ and one observed transition per participant with noise standard deviation 0.5, the lower 95% confidence bound on the gain exceeds 0.770 with probability 0.80 at 593 participants by the closed-form power calculation. Simulated trials of that size reach the conclusion with probability 0.798, against 0.13 with 50 participants and 0.90 with 800 (Figure 3b).

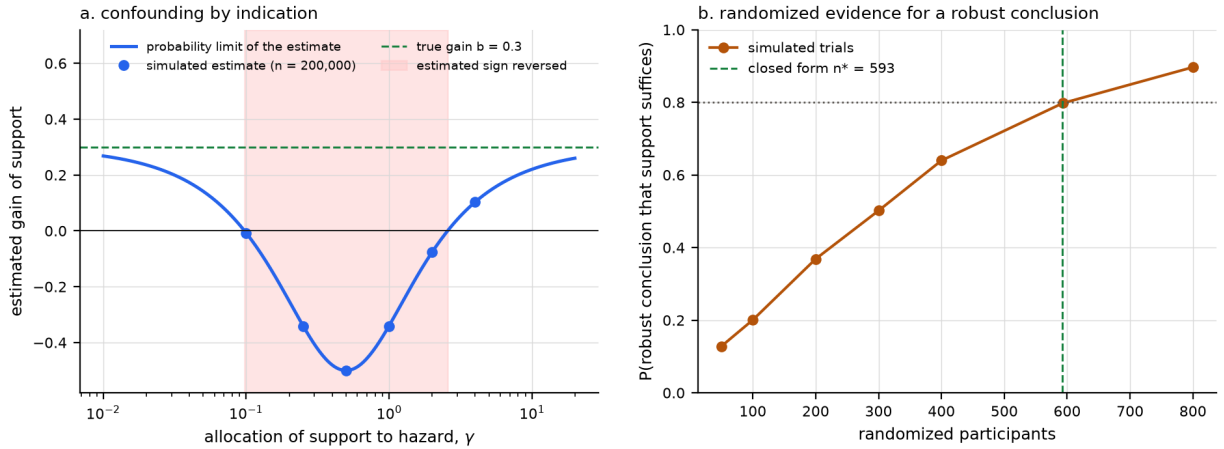


Figure 3: Identification of the effect of support. (a) The estimated gain under confounding by indication against the strength γ with which support is allocated to an unobserved hazard; the shaded interval is where the estimated sign is reversed. (b) Probability that a randomized trial supports the robust conclusion that support of bound 0.5 suffices, against the number of participants, with the closed-form sample size.

Coordinates and distance to recovery

Distances in state space depend on how the state is measured. Consider a two-dimensional state made of the recovery coordinate x and the support available, and measure the Euclidean distance

from each of 400 synthetic individuals to a target at $x = 0.5$ and full support, with the support axis weighted by w to represent a choice of units. Across w from 0.01 to 100, the AUROC of distance for predicting recovery by a horizon of 20 ranges from 0.63 to 0.96, and Kendall's rank correlation between distance and hitting time, among those whose hitting time is finite, ranges from 0.24 to 0.69 (Figure 2c). The hitting times and the set of people who recover are unchanged across all these choices, because they are properties of the dynamics and are preserved by any smooth invertible change of coordinates, which maps trajectories to trajectories. A claim that one person is "further from recovery" than another is therefore meaningful only relative to a stated measurement model, while claims about reachability, hitting probabilities and minimal times are not. Statements about curvature or distances in a psychological state space need the same qualification.

Instruments such as the Clinician-Administered PTSD Scale for DSM-5 (Weathers et al. 2018) and the International Trauma Questionnaire (Cloitre et al. 2018) provide the observation model that any such state must be estimated through. Large prospective cohorts that follow trauma survivors from the emergency department, such as AURORA with a target sample of 5,000 followed for one year (McLean et al. 2020), supply the kind of repeated measurement needed to estimate transition dynamics, but not the randomized variation in support needed to identify its effect.

Limitations

The results concern a one-dimensional bistable model with additive, constant-gain support. Real symptom dynamics are multidimensional and nonstationary, the gain of an intervention varies across people and over time, and a person's capacity to receive support is itself affected by symptoms. The threshold, bottleneck and effort results hold for this model and its local neighbourhood of models with a single saddle-node; how the relevant thresholds are shaped in higher dimensions depends on the structure of the escape path. The synthetic populations are constructed so that support varies independently of severity, which fixes the size of the severity result; in clinical populations the two are correlated and the incremental value of accessibility must be estimated. The confounding model is linear and Gaussian. None of the results is evidence about any treatment, and the model provides no basis for withholding or delaying established care. The notion of a "path" in this paper refers to objective reachability under the model; the perceived availability of pathways, which hope theory treats as a distinct construct (Snyder 2002), and a person's knowledge of which actions make progress are separate quantities that the model does not represent.

Conclusion

A geometric description of recovery is useful to the extent that its quantities are invariant and identifiable. In the canonical bistable model with bounded support, three kinds of statement meet that standard: whether recovery is reachable, how long it takes and how much effort it needs, and how noise converts a threshold into a probability. Distances to recovery in state coordinates do not. None of the reachability statements can be estimated from untreated data, and observational data in which support follows need can reverse the estimated effect of support; randomized evidence

of a computable size is required before a model of this kind supports a conclusion that a given level of support suffices.

Reproducibility

The simulation, its three figures and every model number quoted above regenerate from one command in the repository (`simulation/run_all.py`, seeded). Twenty-one invariants fail the run if broken, among them the agreement of the threshold with a grid maximisation, the saddle-node exponent and asymptote, the agreement of quadrature with Runge–Kutta integration, the monotone decrease of dose with intensity, the closed-form minimal effort, the agreement of simulated and Kramers probabilities where recovery is rare, the closed-form confounding bias against simulation, and the closed-form trial size against simulated trials.

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